Amendments to the Specification:

Please replace the paragraph at page 2, lines 3-13, with the following paragraph:

The instant invention represents a significant improvement over current technologies for probing transmembrane receptor function. Current methods for identifying activating for receptors ~~is~~ are, at best, medium throughput (*See* Stadel, *et al., supra.*). Elaborate and sophisticated molecular assays designed to measure such parameters as calcium mobilization, cAMP, GTP-γS binding, inositol phosphate production, MAP kinase activation, *etc.*, are used to identify activating substances. These assays involve either sophisticated machines that can detect receptor activation in transgenic mammalian cell lines (Sullivan, *et al., Methods Mol Biol* 114: 125-33 (1999)) or labor-intensive methods, such as microinjection of *Xenopus* oocytes followed by electrophysiological recording (Wagner, *et al., Cell Physiol Biochem.* 10(1-2): 1-12 (2000)) Such assays involve direct mechanical detection of receptor function.

Please replace the paragraph at page 4, lines 7-13, with the following paragraph:

During the practice of this method, wherein the known phenotype is selected from the group consisting of: exploded (Exp), dumpy (Dpy), long body (Lon), hyperactive movement (Hpr), paralyzed (Prl), molt defect (Mlt), sterile (Ste), sick (Sck), body morphology defect (Bmd), vulvaless (Vul), slow growth (Gro), egg laying defect (Egl), larval arrest (Lva), ~~larval lethal (Let),~~ protruding vulva (Pvl), multiple vulva (Muv), sterile progeny (Stp), small (Sma), clear (Clr), blistered (Bli), high incidence of male progeny (Him), roller (Rol), larval lethal (Lvl), uncoordinated (Unc), embryonic lethal (Emb).

Please replace the paragraph bridging page 15, line 31 through page 16, line 11, with the following paragraph:

The ability to reprogram the chemosensory response of C. elegans is not limited to misexpressing C. elegans receptors. At a recent C. elegans worm ~~meeting (Tobin, et al., West Coast Worm Meeting, June 23-26, 2000; http://elegans.swmed.edu/WCWM/2000/)~~, a second example of reprogramming the chemosensory response of C. elegans was presented using a mammalian-derived receptor. Summary of Tobin, *et al., Neuron* 35: 307-318 (2002). Expression of the mammalian capcaicin receptor VR1 in sensory neurons of C. elegans conferred a chemoavoidance behavior in response to capsaicin. Like the odr-10 example discussed above, this report is a demonstration that nematode chemosensory behavior in

response to substances can be modified by expression of a receptor in the sensory neurons of nematodes. Unlike our invention, the authors do not express the human capsaicin receptor in C. elegans with the purpose of using chemotactic behavior as a way of identifying substances that activate the receptor, nor do they propose that such an application is possible. We propose that the modification of chemosensory behavior by human 7TMR expression in sensory neurons can be applied to the identification and characterization of substances that activate human 7TMRs or modify human 7TMR activity.

Please replace the paragraph at page 31, lines 10-27, with the following paragraph:

Large-scale functional evaluation of the *C. elegans* genome has defined a number of standard phenotypes that are easily scored by trained *C. elegans* biologists: exploded (Exp), dumpy (Dpy), long body (Lon), hyperactive movement (Hpr), paralyzed (Prl), molt defect (Mlt), sterile (Ste), sick (Sck), body morphology defect (Bmd), vulvaless (Vul), slow growth (Gro), egg laying defect (Egl), larval arrest (Lva), ~~larval lethal (Let),~~ protruding vulva (Pvl), multiple vulva (Muv), sterile progeny (Stp), small (Sma), clear (Clr), blistered (Bli), high incidence of male progeny (Him), roller (Rol), larval lethal (Lvl), uncoordinated (Unc), embryonic lethal (Emb) (Maduro, *et al.*, *Genetics* 141, 977-88 (1995); Piano, *et al.*, *Current Biology* 10, 1619-22 (2000); Gonczy, *et al.*, *Nature* 408, 331-6 (2000); Fraser *et al.*, *Nature* 408, 325-30 (2000)). Of these phenotypes, four can directly result from modification of nervous system function by gene mutation or expression of modified proteins: Hpr, Prl, Egl, and Unc. For example, expression of activated G proteins in the nervous system can lead to an Egl phenotype; in addition, mutations in a number of human 7TMR signaling pathway proteins can lead to an Egl phenotype, or suppress the action of a second mutation that leads to the Egl (Wilkie, *Current Biology* 10, R853-6 (2000)). Therefore, expression and activation of a human 7TMR could perturb the *C. elegans* nervous system and manifest or modify these phenotypes. The phenotypic read-out could then lead to evaluation of substances that would alter human 7TMR activation.